THE VETERINARY RECORD

FOUNDED BY WILLIAM HUNTING, F.R.C.V.S., 1888

No. 3. JANUARY 15, 1949 VOL. 61.

Epileptic States in the Dog, with Special Reference to Canine-Hysteria *

By

HERBERT B. PARRY

ANIMAL HEALTH TRUST, KENNETT, NEWMARKET

Epilepsy has been known from prehistoric times and in man has become the subject of a vast literature. Even to-day it is one of the major chronic disabling diseases. Among the lower mammals the condition is not common except in the dog, which is peculiarly liable to the disorder, a fact which has been recognised for at least two centuries (Anon., 1735). This special incidence in the dog makes the problem one of peculiar interest, for one immediately asks, why should the brain of the dog be so liable to such dysfunction while the brains of other species are not? Perhaps if we can lay bare some of the reasons why the dog should be so prone to epilepsy we may shed some light on the much vaster field of cerebral organisation, and the cause of brain dysfunction of many kinds.

Epilepsy was known in ancient times as the "falling sickness" since the major convulsions now known as grand-mal seizures were thought to be the principal feature of the disease. However, we now recognise many other clinical manifestations and it is convenient to refer to this whole group as "epileptic states" or "the epilepsies." To provide a definition of what is meant by this term is a matter of great difficulty for it represents a symptom-complex rather than a disease entity. Hughlings Jackson attempted a physiological definition and said that epilepsy is the name for "occasional, sudden, excessive, rapid and local discharges of grey matter" (Taylor, 1931). However, it is now known that such discharges do occur in conditions other than epileptic states. Kinnier Wilson (1940) declined to attempt to define the disorder. However, for our purpose I feel that we should attempt some definition, which may be used in attempting to clarify the problem in the dog, however tentative it may be.

We may define epileptic states as transient disturbances of brain function, which may or may not give rise to abnormal motor, emotional and social behaviour, and are usually accompanied by some disorder of consciousness. Such disturbances may or may not be associated with definite morphological changes; in many there are no morphological changes whatever. In fact we may consider epileptic states as the clinical manifestation of dysfunction of the brain which may be elicited by a wide variety of aetiological agents, in the same way as jaundice may represent dysfunction of various aetiology, or the hypocalcaemic states represent a symptom-complex occurring in a number of different diseases.

This hypothesis of the essential unity, in the sense of the common nature of their underlying dysfunction, of a wide variety of clinical disorders which are so often merely termed "fits," is the main theme of my talk to you this afternoon. It is no new idea, for prior to the 1890's these disorders were described as epilepsy. The term "hysteria" was introduced quite casually by Woodroffe Hill (1892) and has since obtained wide acceptance in this country, but not elsewhere. Its chief merit is its wide acceptance, but it should not be confused with "hysteria" in man, which is a purely psychological disorder, whereas hysteria in the dog is an epileptic disorder, usually associated with diet. To prevent confusion I would suggest that the canine disease be always referred to thus: "canine-hysteria."

I wish to discuss firstly some of these epileptic states; secondly, to attempt to show how the syndromes known as canine-hysteria may be fitted into this group; and thirdly, to draw attention briefly to other syndromes, which may be confused clinically with epileptic states. My object is to enlist your co-operation in throwing some light into this dark confused prospect of veterinary medicine.

NATURE OF THE DYSFUNCTION

In the normally functioning central nervous system the balance of activity within individual or groups of neurones is finely integrated, so that the afferent impulses from within and without the body impinging on those neurones evoke only efferent responses of a kind and of a magnitude which we have come to accept by long experience as a normal behaviour pattern. It is when this fine balance is disturbed that we get behaviour patterns so unusual that we say that an animal is having a "fit." The magnitude of the disturbance may be such that a motor convulsion with loss of consciousness ensues; it may be so slight that the only sign is a momentary arrest of the conscious stream—the "absence épileptique" of the French—which one sees in the automatic behaviour stage of canine-hysteria.

The precise nature of the functional defect is almost certainly extremely complex, but its existence may be demonstrated by alterations in the rhythmic electrical activity of the brain as recorded by the electro-encephalograph. These alterations may be termed "dysrhythmias" and such dysrhythmias can be detected when no clinical signs of the malady are apparent; in fact they may be demonstrated in subjects which will never show clinical signs except under the most extreme stress. On the other hand, the failure to detect any such dysrhythmia does not exclude the possible presence of an epileptogenic defect. Knowledge of the electro-encephalogram is of course most highly developed in man, but there are many indications that the epileptogenic defect in the dog is accompanied by a similar brain dysrhythmia. In 1942 I demonstrated such a dysrhythmia in a case of idiopathic "grand-mal" seizures in a red setter (Parry, 1942) and American workers have demonstrated similar changes in dogs fed agenised flour (Newell *et al.*, 1947).

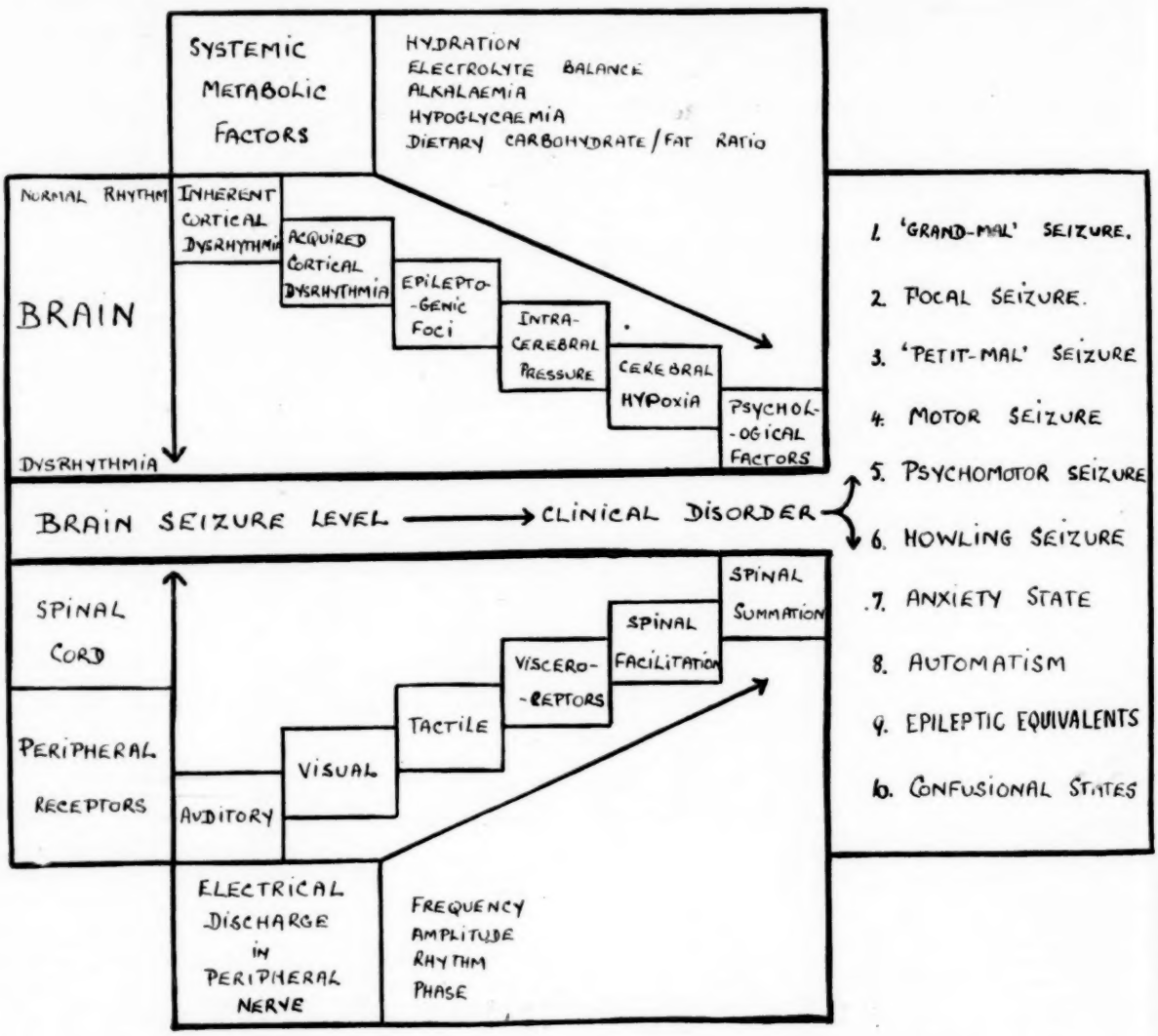
An essential feature of the problem is that whereas the epileptogenic functional defect is continuous in certain cases as revealed by the electro-encephalogram, the clinical expression is irregular and discontinuous, epileptic episodes occur only when various circumstances summate to exacerbate the dysrhythmia to a level such that a brain seizure of some kind occurs. When the dysrhythmia has reached this level within a group of neurones, which may be conveniently termed the "brain seizure level" it is probable that a wave of electrical discharge suddenly spreads over surrounding nervous structures, obliterating momentarily their normal function and imposing instead, temporarily, an alien function. Such obliteration of normal function and imposition of an alien function gives rise to the clinical signs which we recognise as a "fit." The precise clinical expression will depend upon the locus of the discharging focus and upon the intensity and extent of the discharge. Thus a slight discharge may merely set up a localised myoclonic seizure, say of the jaws, while a discharge spreading all over the cerebral cortex may give rise to the tonic-clonic convulsion of a "grand-mal" seizure.

FACTORS INFLUENCING THE "BRAIN SEIZURE LEVEL"

There are very many factors which can act in consort to build up the brain seizure level, and thereby evoke an epileptic episode. In the dog they are but imperfectly defined, but we may attempt in a tentative manner to classify them and in due course, I hope, to reduce them to a quantitative basis. It is convenient to consider them under three heads; firstly, factors which affect the response of the brain to afferent impulses arising in various parts of the peripheral sensorium; secondly, factors affecting the nature of the afferent impulse before it reaches the brain; and, thirdly, systemic metabolic factors. These factors have been set out schematically in Fig. 1.

* The substance of this paper was delivered as an address to the Central Veterinary Society on May 6th, 1948. It is intended to be complementary to the paper on "Canine-Hysteria in Relation to Diet"—see *Veterinary Record*. **60.** 389.

Fig. I.—Factors Underlying the Brain Seizure Level and its Clinical Expression.



A Diagram to illustrate the way in which various factors may summate to reach the brain seizure level and the varying types of clinical disorder which may ensue when this level is exceeded.

A. Factors Affecting the Brain's Response

1. Inherent Brain Rhythms

Since we have seen that the essential basis of an epileptic discharge is dysrhythmia sufficiently severe to exceed the brain seizure level, the further the normal brain rhythms are from this level, the wider the functional reserve of the brain before it shows insufficiency, *i.e.*, an epileptic episode: in the same general analogous manner the functional reserve of the heart and circulation has to be reduced before functional insufficiency occurs and clinical signs of circulatory failure ensue. We have at the moment no means of assessing the functional reserve of the brain except by the gross assessment of dysrhythmia, which is a very crude way of doing so. There is thus every opportunity for wide variation between the brain rhythms of different individuals and hence of their liability under similar conditions to exhibit epileptic episodes. Although there has been much talk and speculation with regard to alleged breed differences in liability to epileptic states, there is as far as I am aware no scientific evidence bearing on this point.

2. Inherent Cortical Dysrhythmias

This group of epilepsies, the so-called idiopathic epilepsies, is well recognised in man and has been subjected to much electroencephalographical investigation; in these patients there is an inherent dysrhythmia of the cerebral cortex which renders them liable to spontaneous epileptic episodes. It has been said that this type does not occur in animals but I believe that this condition does occur in dogs. I have seen one case in Sydney, whose E.E.G. showed during the periods free from clinical signs just those changes that are found in man (Parry, 1942). However, much more work is necessary before one could speak with confidence on this point, especially as hitherto the differentiation of the condition from other epileptic states has been so confused.

3. Acquired Cortical Dysrhythmias

During the last few years a great deal of evidence has been accumulated to show that cortical dysrhythmias may become acquired permanently. The commonest cause in man is head injury such as occurs in motor accidents, gun-shot wounds and trauma at birth, which sets up a localised epileptogenic focus. The other main cause is intoxications of various kinds, of which that due to agenised proteins is the most important from our point of view.

(*a*) *Intoxications*

Recent studies have demonstrated that on feeding agenised wheat proteins to dogs cerebral dysrhythmia of varying severity can be rapidly induced (Newell *et al.*, 1947). It seems almost certain therefore that the cardinal effect of agene intoxication in dogs is to induce a cortical dysrhythmia and it is hard to believe that this dysrhythmia is not intimately concerned with the hysterical episodes in view of the evidence in man incriminating similar dysrhythmias with epileptic episodes. We shall return to this subject later.

It is probable that agenised proteins are not the only agents which may induce such dysrhythmia in the dog and there is circumstantial evidence that other dietary regimens and overdosage with certain drugs may also do so.

(*b*) *Localised Epileptogenic Foci*

(i) *Traumatic Injuries*

The essential requirement seems to be the production of scar-tissue between the meninges and the cerebral cortex, while the vascularity of the lesion is also an important factor (Forster & Penfield, 1930). In man these are well recognised and Steel from the Sydney School has recently reported on a case in a dog in which the scar was removed by surgical intervention (Steel & Wyke, 1947).

(ii) *Post-infective Residua*

There are suggestions, particularly from studies in man, that residual damage to the nervous system following an infection involving the central nervous system, *e.g.*, an encephalitis due to a neurotropic virus, in which the inflammatory process has subsided, may act as an epileptogenic focus. Whether such residua actually occur in the dog remains to be proven, but the idea would serve to explain many puzzling clinical cases.

(iii) *Other Small Lesions*

These include small cysts and small subdural haematomata.

4. Intra-cerebral Pressure

There is much evidence to support the contention that an increase of the intra-cranial pressure, be it due to the cerebral oedema of certain entero-toxaemias or of lead encephalopathy, or to space-occupying lesions of the cranium, such as large cysts, large haematomas, tumours and abscesses, can greatly increase the liability to epileptogenic episodes. The important effect is probably an increase in intra-neuronal pressure, for there is considerable evidence that increased fluid retention by the brain is an important precipitating factor in canine-hysteria (Parry, 1948a, b).

5. Psychological Factors

We know very little of the role purely psychological factors play, but it is established that the mere exposure of an hysteria-prone animal to a situation or environment which has by previous experience acquired a strong emotional content for the animal, *e.g.*, the scene of a fight or unlooked-for reprimand, may precipitate an attack. In man an epileptic-prone patient—frustrated, bored, and with his mind not fully occupied—is much more liable to episodes than the same patient fruitfully occupied mentally.

B. Factors Affecting the Nature of the Afferent Stimulus

Since the precise nature of the afferent stimulus as it reaches brain neurones, its frequency, rhythm, amplitude, etc., may determine whether the brain seizure level be reached or not, factors which influence the afferent stimuli may be of considerable importance.

1. Peripheral Receptors

Stimuli arising in certain peripheral receptors seem to be more liable to precipitate episodes than do others. In the dog auditory stimuli are peculiarly liable to do so, a fact well recognised in hysteria-prone dogs, in which the sound of a jet of water suddenly released or the bark of an hysterical dog may be potent precipitating agents (Parry, 1948b). It is of interest in this connection, that a highly inbred strain of mice has been developed in which fatal epileptic convulsions can be induced at will by sounds of suitable note and intensity, such as rattling a bunch of keys or the discharge of an electric bell (Hall, 1947). Suitable stimulation of other peripheral receptors can also be important although precise study and controlled stimulation has not been attempted yet in the dog. However, there seems no reason why alimentary helminths should not stimulate the enteroceptors, and the association of acariasis or foreign bodies in the external auditory meatus with canine-hysteria, so stoutly propounded by the early French workers (Mégnin, 1881; Nocard, 1882), are almost certainly examples of suitable stimulation of skin receptors precipitating epileptic episodes. Cases are on record of peritonitis being the exciting cause (Steel, 1948).

2. Nature of the Stimulus

It is not possible to define at all clearly at the present time what stimuli will and what stimuli will not evoke epileptic episodes. Indeed it is likely that they will vary with the brain dysrhythmia which is the prime cause. In general one can only say that stimuli which produce in afferent nerve pathways electrical discharges of a frequency, wave-form and amplitude likely to be readily superimposed on the brain rhythms, and, if in phase, to lead to rapid summation, are most likely to cause the brain seizure level to be exceeded.

3. Spinal Cord and Afferent Pathways

The role of these structures is at present purely a matter of surmise, but any state in the cord which is likely to increase the ease with which peripheral impulses are transmitted or enlarge their central termination, may well increase the importance of peripheral stimuli as precipitating agents. Whether the spinal cord can act as a primary epileptogenic focus in the dog is not known.

C. Systemic Metabolic Factors

In man a number of systemic metabolic factors are known to play a quite considerable role in the occurrence of epileptic motor episodes, and these factors are known to exert a similar influence in experimental canine-hysteria due to agenised wheat gluten (Parry, 1948a, b). It seems likely, therefore, that they may influence other epileptic states in the dog. Thus water retention, especially if accompanied by a low sodium chloride intake, can be made, if pressed to extremes, to precipitate seizures (Allen, 1945). In fact, it is virtually impossible to produce hypoglycaemic convulsions in the dog without also ensuring a considerable degree of water retention together with a raised cerebro-spinal fluid pressure (Drabkin & Ravdin, 1937). On the other hand, a high-fat-low-carbohydrate diet probably suppresses the motor episodes, while I have evidence that the thiamine saturation in the dog may be important (Parry, 1948a, b). Similarly the alkalaemia of hyper-ventilation may precipitate episodes in man. Disturbances of acid-base balance may well explain the isolated episodes observed in gun-dogs towards the end of a hard day's work, particularly if they are not physically fit.

4. Cerebral Hypoxia

Mackenzie first drew attention to the fact if the cerebral circulation was interrupted for as long as 16 seconds by some cardiac emergency such as heart block, the patient would exhibit a convulsive seizure with loss of consciousness. Defective oxygen supply to the brain due to a variety of circulatory disorders, such as vasovagal syncope and paroxysmal tachycardia, do increase the tendency to epileptic episodes in epileptic-prone subjects (Penfield & Erickson, 1941). It is perhaps pertinent to enquire whether or not some of the "fits" seen in animals with encephalitis associated with perivascular oedema and cuffing are not due to relative hypoxia of the neurones following interference with the diffusion of oxygen into the brain substance by the perivascular lymphocytic infiltration and oedema.

THE CLINICAL MANIFESTATION OF THE "BRAIN SEIZURE LEVEL"

The clinical signs of the epileptic discharge depend on three things, firstly, its originating locus within the central nervous system, secondly, the nature of the epileptogenic focus—whether it is sharply demarcated, *i.e.*, focal, or diffuse—and thirdly, the severity of the discharge, *i.e.*, the amount of brain which is

involved and the degree of interference with the functioning of the parts involved.

In assembling the clinical data upon which it is possible to deduce with considerably accuracy the precise locus and focus of the discharge, we are at a peculiar disadvantage compared with human physicians, since so many of the clinical signs are purely subjective and are related to alterated sensibility, unusual feelings and disturbances of memory, of which we have no means of knowing in animals. We are thus forced to rely almost entirely upon disturbances of motor function aided by any observable behaviour changes. As far as we know at present, by far the most common locus in dogs lies in the cerebral cortex, giving rise to motor seizures of one kind or another. In man epileptogenic foci occur in the purely sensory areas of the cortex, such as the visual and temporal lobes; in addition, so-called "diencephalic" epilepsy, in which the focus is probably in the hypothalamus or thalamus and whose clinical signs are essentially those of disturbances of the vegetative nervous system, is recognised. Cerebellar seizures occur when the focus is in the cerebellum.

Our knowledge of the locus of primary epileptogenic foci in animals is still so small that it is not possible to relate the clinical pattern with any given locus, and I shall only attempt to describe the main clinical types.

1. *"Grand-mal" Seizure.*—There is a short initial aura, most commonly of retraction of the head with champing of the jaws, which is probably indicative of wide cortical representation of mouth and jaw movements due to the special importance of these movements for self defence and gathering food in the dog. This stage is followed by generalised tonic spastic extension of all parts of the body with loss of postural control, cessation of respiration and complete loss of consciousness. The tonic phase with the animal lying on its side usually lasts some 15 to 30 seconds when it passes into the clonic phase, during which respiration is resumed and the tonic extension of the body is replaced by co-ordinated running movements of the limbs. This phase may last one minute or longer. During the seizure there is loss of control of the sphincters; there is evacuation of faeces and incontinence of urine, while saliva drools form the mouth. On recovery the animal appears to have no recollection of events during the seizure.

2. *Focal (Jacksonian) Seizure.*—In these there is a definite initial focal sign, such as the twitching of a limb, the jaw, a group of facial muscles, etc., which is usually unilateral only and lasts several seconds and is followed immediately by the generalised "grand-mal" type of seizure, in which the animal falls on one side, the side of the lesion. The interest of these focal seizures lies in the clue which they provide as to their aetiology, for the epileptogenic focus will be in that part of the cerebral cortex which represents the part of the body involved in the initial focal sign. These seizures are nearly all due to some acquired dysrhythmia, most commonly a depressed fracture of the cranium (Steel & Wyke, 1947).

3. *"Petit-mal" Seizure.*—In these seizures the stream of consciousness is disturbed, but is not usually interrupted completely, so that the animal appears to be unaware of its immediate environment but not unconscious. There are usually motor signs such as myoclonic movements of small groups of muscles, especially of the head and neck, but postural control is usually retained. Often these seizures are so slight as to escape notice unless the dog be under the closest observation; others may be so severe as to resemble "grand-mal" seizures except that the animal does not fall over unconscious. Normally a "petit-mal" seizure lasts but a few seconds.

4. *Motor Seizure.*—These show loss of postural control so that the animal falls over on its side, where it exhibits violent paddling movements of the limbs, which are often unco-ordinated in the sense that the sequence of movements does not follow those of normal gaits and are often unsymmetrical between the two sides. Consciousness is still present, but there is usually some departure from the normal.

5. *Psychomotor Running Seizure.*—This is the well-known "running fit" of canine-hysteria. Postural equilibrium is excellent and the gait normal, but the animal runs about very rapidly as if "chased by the devil." Peripheral vision is not impaired, but the perception of visual impressions may be suppressed, so that the animal may run into minor objects, but does not collide with solid obstructions. Consciousness is disturbed and distorted by the signs of acute anxiety and apprehension, which accompany the running. The animal is not fully cognisant of its surroundings and in severe seizures appears to have no recollection of events occurring during them, for dogs will run for miles and become completely lost, apparently having no idea of the route they have taken. Control of the sphincters is not lost, but drooling of saliva from the mouth may occur.

6. *Howling Seizure.*—The animal sits and howls in an unusual key, either high-pitched or in the bass. No amount of intimidation will persuade him to desist. Occasionally these seizures occur alone, but more usually they occur with the running seizure as a "running barking fit."

7. *Anxiety States.*—In these states the animal shows apprehension of some unknown vaguely comprehended situation and acts as if very frightened. The animal will not play, and walks around with its ears laid backwards, tail carried between its legs, casting furtive glances behind it as it goes, and often hiding in the darkest, most inaccessible places. Similar signs of anxiety can occur in timid animals following brutal treatment, but these anxiety states occur spontaneously in animals in which there has been no mal-treatment of any kind.

8. *Automatism.*—This is a state in which the animal is seemingly fully conscious of its surroundings, has normal control of posture and movement, and yet is not aware of its surroundings, *i.e.*, its pattern of behaviour is determined entirely by previous experience and is not influenced at all by current appreciation of its immediate situation. An animal in this state behaves like an automaton, but so closely does its behaviour simulate the normal that this state is frequently overlooked.

9. *Epileptic Variants or Equivalents.*—This term is given to certain border-line cases of human epilepsy, who have abnormal E.E.G.'s and who exhibit no gross disturbances of consciousness or of motor function, but in whom relatively minor alterations of brain function occur for short periods. There are three main types (Pugh, 1947); firstly, motor variants in which there is aggressiveness and violence or emotional imbalance with loss of memory; secondly, sensory variants in which there may be illusions, altered skin sensibility and pain with partial loss of memory; and, thirdly, autonomic variants in which disturbances of sympathetic and parasympathetic function occur, causing, for example, transient vasomotor disturbances such as tachycardia, cyanosis, etc. These variants have not been definitely recognised in the dog, but their differentiation, especially of the motor variants, from canine-hysteria is a matter of great difficulty, and the possibility of their occurrence must be considered.

10. *Confusional States.*—Into this category, which is only included tentatively, one can place those cases of exaggerated response to normal handling or situations, such as exaggerated fighting against restraint in a usually docile animal, and outbursts of "blind rage" provoked by normally innocuous situations. There may be loss of postural control but consciousness is unimpaired.

SYNDROMES INVOLVING EPILEPTIC STATES

The various manifestations of epileptic states are met with clinically as components of a wide variety of disorders in the dog. It is only possible to mention briefly the disorders in which signs of the epileptic states occur most commonly.

A. *Idiopathic Cerebral Dysrhythmias (Idiopathic Epilepsies).*
   1. "Grand-mal" epilepsy.
   2. "Petit-mal" epilepsy.
   3. Epileptic variants or equivalents.

B. *Cortical Focal Seizures (Jacksonian Epilepsy).*
   1. Depressed fractures of the cranium.
   2. Cortical scar formation.
   3. Small sub-dural haematoma.
   4. Post-infective residua.

C. *Canine-hysterias (Hysterical States).*
   1. Dietary
      (*a*) Wheat gluten canine-hysteria.
      (*b*) Agene intoxication.
      (*c*) Other non-wheat canine-hysterias.
   2. Miscellaneous
      (*a*) Auricular acariasis.
      (*b*) Arctic dog disease.
      (*c*) Eclamptic seizures.

D. *Infective Syndromes (Neurotropic Viruses).*
   1. Prodromal Laidlaw-Dunkin distemper.
   2. Acute primary 'encephalitis.'
   3. Post-septicaemic secondary 'encephalitis.'

E. *Syndromes of Increased Intra-cerebral Pressure.*

1. Increased cerebro-spinal fluid pressure.
2. Cerebral oedema:
   (*a*) Lead encephalopathy.
   (*b*) Hypertensive states.
   (*c*) Entero-toxaemias.
3. Space-occupying lesions:
   (*a*) Tumours.
   (*b*) Abscesses.
   (*c*) Haematomata.

This list must be considered as tentative only, designed to clarify the investigation of these nervous disorders of the dog.

In many cases the requisite information to establish a definite diagnosis is not available for the dog, and data from other species have therefore been included.

A. *Idiopathic Cerebral Dysrhythmias (Idiopathic Epilepsies)*

The clinical expression of these dysrhythmias is not diagnostic; the signs are those of the grand-mal seizure, petit-mal seizure, etc., which occur in a number of different aetiological entities. The diagnosis of these dysrhythmias depends, therefore, in the first place, on excluding other possible causes, *e.g.*, canine-hysteria, but ultimately the presence of the cerebral dysrhythmia must be established by electro-encephalographic means. As far as I am aware, the only case in which this has been done was the Sydney case noted above (Parry, 1942). Confirmatory evidence is the failure to demonstrate, using adequate methods, any lesion in the brain which might act as an epileptogenic focus.

B. *Cortical Focal Seizures (Jacksonian Epilepsy)*

It is often possible by a very careful evaluation of the clinical signs exhibited at the onset of such a seizure to obtain a useful guide as to its cause and the location of the epileptogenic focus (Steel & Wyke, 1947). A careful enquiry may often elicit some history of trauma, etc., which may be confirmed by clinical inspection, X-ray examination and ventriculography. Cortical scar formation and post-infective residua may only be shown on careful examination after death, although E.E.G. records may prove of value as they have done in man.

C. *Canine-hysterias (Hysterical States)*

Since as far as we know at the moment the essential aetiological factor in the first sub-group is some dietary defect, most usually agenised wheat products, the recognition of this group must depend on the occurrence of some recognised hysteriagenic agent in the diet being ingested in amounts sufficient to exceed the maximum tolerated daily dose (Parry, 1948, b). If the suspected hysteriagenic agent be removed by changing the diet, the condition should subside within a few days, leaving no residual neurological defects, except in very severe cases in young puppies. In fact, if there are focal neurological signs in adult dogs, the disorder is almost certainly not a canine-hysteria, especially if the defect is asymmetrical between the two sides of the body. Similarly examination of the body after death will reveal no lesions whatever in the central nervous system, *i.e.*, the dysfunction in this group is purely of a functional nature and completely reversible. In the second sub-group we may place conditions such as Arctic dog disease (Baashuus-Jensen, 1935) and the auricular acariasis of the French (Mégnin, 1881; Nocard, 1882), the clinical accounts of which are indistinguishable from what we now know as dietary canine-hysteria, but in the aetiology of which the role of diet is not clear, although one thing is certain—there was no question of agene intoxication being present.

D. *Infective Syndromes due to Neurotropic Viruses, etc.*

Any discussion of this group must for the moment remain entirely provisional until a detailed account of the work briefly reported by MacIntyre, Trevan and Montgomery (1948) is available. Nor is this the place to enter into any detailed consideration of this difficult subject. Suffice it to draw attention to three or four clinical entities, whatever their aetiology proves to be. During the prodromal stage of infection with what on clinical grounds appears to be Laidlaw-Dunkin distemper, it is not uncommon to see a dog suffering from a clinical syndrome identical with that of dietary canine-hysteria. The animal may be on a diet of low hysteriagenic potency and thus be in an aclinical state of hysteria-proneness (Parry, 1948a, b), but the association with the development of clinical signs of distemper with catarrh of the eyes, nose and alimentary tract some 48 hours later does suggest that the virus infection during its septicaemic phase can act as an epileptogenic (or hysteriagenic) factor.

The second sub-group, the acute primary encephalitides, embraces those cases in which encephalitic signs develop during the initial phase of the illness immediately following the febrile reaction. The encephalitic signs embrace a wide variety of neurological signs, often of a focal character; but not infrequently "grand-mal" seizures occur, and may be so severe as to be almost continuous. Whether these epileptic signs are due to definite invasion of the neurones by the virus, or whether they are due to secondary effects of the virus on the oxygen supply to the brain, *i.e.*, by causing perivascular lymphocytic infiltration and oedema, is not known. However, in these cases it is wise to look for other signs of circulatory insufficiency to the brain before assuming that irreparable damage to neurones has occurred.

The third group, the post-septicaemic secondary encephalitis cases, includes many of those cases often referred to as "post-distemper" fits, *i.e.*, they occur some days or weeks after an attack of a febrile illness and when the signs associated with the febrile phase have abated or disappeared. In many instances seizures of a grand-mal type occur with considerable frequency prior to death. The brains of these cases show generalised damage to the higher brain centres (Perdrau & Pugh, 1930; Hurst, Cooke & Melvin, 1943), *i.e.*, it is not possible to decide whether there has been any specific epileptogenic focus, either from the clinical or pathological evidence, since the damage is so widespread.

E. *Syndromes of Increased Intra-cerebral Pressure*

In this group we can place for the time being a number of seemingly unrelated observations, which connect sporadic epileptic cases with increased intra-cerebral pressure.

Of the importance of the cerebro-spinal fluid pressure in determining the occurrence or otherwise of grand-mal type convulsive seizures little evidence is available from the clinical literature, but there is some very illuminating experimental data. While investigating the mechanism of hypoglycaemic convulsions in the dog produced by large doses of insulin, Drabkin & Ravdin (1937) found that they were unable to induce convulsions in intact animals unless the cerebro-spinal fluid pressure was above 150 mm. of water. The sporadic convulsions which occur in vitamin A-deficient animals (Madsen, 1940; Jones, Schmidt *et al.*, 1943) in which the C.S.F. pressure is known to be raised (Moore & Sykes, 1946) may well be related to the increased C.S.F. pressure.

The role of cerebral oedema is not well defined in the dog, but evidence from other species suggests that it must be considered in the dog. Thus the acute lead encephalopathy of cattle is associated with increased C.S.F. pressure such that severe choking of the optic disc occurs (Parry, 1941). In man the cerebral oedema accompanying arterial hypertension is not infrequently accompanied by epileptiform convulsions (Alajouanine, 1947), while the convulsions which are seen in the entero-toxaemia of sheep due to *Clostridium welchii* type B are almost certainly due to the neurotoxic effect of the toxin in producing cerebral oedema (Kellaway *et al.*, 1940).

Association of epileptic seizures with space-occupying lesions such as tumours, abscesses and haematomata are recognised in man; that they may be responsible for occasional cases in the dog seems not unlikely, although lesions of these kinds are very much rarer in the dog than in man.

THE DIFFERENTIAL DIAGNOSIS OF THE EPILEPTIC SYNDROMES IN THE DOG

The differential diagnosis of the various disorders in which epileptic signs may occur is often a matter of very great difficulty, even when laboratory aids are available to supplement clinical observation. Generally only a tentative diagnosis can be made of clinical cases after exhaustive neurological examination of the patient and full knowledge of the history of the case. It is largely a matter of the elimination of possibilities. Hence the paramount importance in experimental studies of these canine epilepsies that every precaution be taken to exclude all possible causes other than those under investigation. In the following scheme an attempt has been made to provide some guidance in arriving at a diagnosis; it is put forward as a very tentative one in the hope that it may help to clarify our ideas on these disorders. We may conveniently divide these disorders into three sections (see Table I).

TABLE I

THE DIFFERENTIAL DIAGNOSIS OF THE EPILEPTIC STATES

Section	Principal signs	Sub-section	Clinical disease
I	Seizures sporadic, generalised and symmetrical No initial focal signs No continuing neurological defects No morphological lesion Loss of postural control	(*a*) "Grand-mal" seizures only	"GRAND-MAL" EPILEPSY
		(*b*) "Grand-mal" seizures with psychomotor anxiety signs	HYSTERICAL STATES 1. Dietary canine-hysteria 2. Agene intoxication 3. Arctic dog disease 4. Auricular acariasis 5. Prodromal distemper hysteria 6. Eclamptic anxiety
		(*c*) Motor seizures with mania	CEREBRAL OEDEMA 1. Lead encephalopathy 2. Entero-toxaemias 3. Hypertensive states
II	Seizures often frequent, generalised, and dysfunction often not symmetrical Initial focal signs Continuing neurological defects present Definite morphological lesions Loss of postural control Gross impairment of consciousness	(*a*) Infective	ENCEPHALITIDES (ENCEPHALOPATHIES) 1. Acute primary "encephalitis" 2. Secondary "post-distemper" 'encephalitis'
		(*b*) Non-infective	CORTICAL INJURIES 1. Depressed fractures 2. Scar formation 3. Sub-dural haematoma 4. Post-infective residue SPACE OCCUPYING LESIONS 1. Tumours 2. Abscesses 3. Large haematoma and cysts
III	Seizures infrequent, local and often inconspicuous Small departures from normal behaviour only Postural control retained Consciousness affected to less degree	(*a*) Slight motor and autonomic disturbances	"PETIT-MAL" EPILEPSY EPILEPTIC EQUIVALENTS
		(*b*) Automatic behaviour	POST-HYSTERICAL AUTOMATISM
		(*c*) Over-reaction	CONFUSIONAL STATES 1. Teething 2. Ascaridiasis

Section I includes the disorders which are in the main of a purely functional nature. The epileptic seizures occur occasionally either singly or in a small series; exhibit no initial focal signs, show symmetrical dysfunction with disturbances of motor function and impairment of consciousness. Once the seizure has subsided there are no continuing neurological defects, *e.g.*, cranial nerve palsies. There are no demonstrable morphological lesions, except in some of the cerebral oedemas, where evidence of the oedema may be apparent in microscopic preparations.

In sub-section Ia the essential clinical sign is the grand-mal seizure alone, due to "grand-mal" epilepsy. In sub-section Ib the grand-mal seizure is accompanied to varying degrees by psychomotor and anxiety signs, any or all of which may be present, and which are seen in the hysterical states including the dietary canine-hysterias. In this sub-section we may also place, on clinical grounds, the hysterical state so often seen during the prodromal period of Laidlaw-Dunkin distemper and the anxiety state seen in suckling bitches and often called post-parturient eclamptic anxiety; the relation of this condition to dietary canine-hysteria is obscure. In sub-section Ic, which differs rather from the two previous sub-sections, the pattern of the seizure is less well defined but usually takes the form of a motor seizure with maniacal behaviour; this sub-section includes the disorders associated with cerebral oedema and increased cerebro-spinal fluid pressure.

Section II is characterised by the non-symmetrical distribution of the nervous dysfunction between the two sides of the body and the presence at the beginning of a seizure of focal signs.

Neurological defects are often present continuously between the seizures and there are definite morphological lesions. The seizures may occur sporadically or in a continuous series. There are two sub-sections, an infective and a non-infective. In sub-section IIa we have the epileptic episodes associated with the primary and secondary 'encephalitides' (or encephalopathies) due to infective agents. In sub-section IIb, the non-infective sub-section, we have the cortical focal seizures due to various injuries to the cerebral cortex, and the much rarer focal seizures seen with space-occupying lesions.

In section III the degree of disturbance of normal function is very much less than in the two previous sections; the episodes are often infrequent and inconspicuous, with often only local disturbances of function. Often these take the form of small departures from normal behaviour patterns; postural control is retained and consciousness may not be affected conspicuously. There are no morphological lesions, nor continuing neurological defects. In sub-section IIIa slight disturbances of motor control and autonomic nervous regulation occur, as in petit-mal and the epileptic equivalents. In sub-section IIIb the principal sign is automatic behaviour, such as is seen in the post-hysterical automatism of canine-hysteria. In sub-section IIIc certain conditions occur in which the essential sign is an abnormally marked reaction to simple situations, such as one observes in the confusional states associated with teething and ascariasis.

SYNDROMES SIMULATING EPILEPTIC STATES

There are a number of conditions in which neurological signs occur clinically and which may on superficial examination be confused with the epileptic states. They fall under five sections, which are set out in Table II.

Section I includes the most important disorders, namely those in which grave interference with the blood supply to a part of the brain occurs. If such interference occurs suddenly, loss of consciousness ensues within a few seconds accompanied in severe cases by

TABLE II
SYNDROMES SIMULATING EPILEPTIC STATES

Section	Principal signs	Nature of disorder	Clinical syndromes
1	Sudden loss of consciousness with motor seizures	(*a*) ACUTE HEART FAILURE	Heart block Paroxysmal tachycardia Vasovagal syncope Sudden massive haemorrhage Sudden coronary insufficiency
		(*b*) CEREBRAL VASCULAR ACCIDENTS (APOPLEXY)	Sudden haemorrhage Sudden occlusion by emboli and thrombi (?) arterial spasm
2	Hypermyotonia without loss of consciousness	(*a*) INTOXICATIONS	Strychnine Tetanus
		(*b*) METABOLIC DISORDERS	Parathyroid insufficiency Hypocalcaemic states
		(*c*) NEUROMUSCULAR DISORDERS	" Scotch cramp "
3	Mania with or without motor seizures	(*a*) DRUG INTOXICATIONS	Fluoroacetate Benzedrine
4	Anxiety and apprehension onlv	(*a*) NON-EPILEPTIC ANXIETY STATES	Neuroses Experimental neurasthenia
5	Inconstant motor seizures	(*a*) RENAL INSUFFICIENCY	" Uraemia "
		(*b*) ECLAMPSIA	Pre-parturient eclampsia
		(*c*) METABOLIC DISORDERS	Hypoglycaemia (inconstant)

motor movements, which may be very similar to an epileptic seizure. In fact in an epileptic-prone subject, the cerebral hypoxia occurring as a result of a failing blood supply may be sufficient to evoke an epileptic episode. However, in such cases the prime cause is circulatory and not neurogenic. Inadequate blood supply to the brain is most commonly due to either acute heart failure with failure of the forward circulation or some cerebral vascular accident producing apoplexy. The more usual causes of these conditions are given in Table II.

In section 2 we have those cases in which consciousness is essentially normal, but in which there are episodes of excessive muscular tone, which may be so severe that the animal loses its postural control and falls to the ground. These episodes of hypermyotonia may proceed to tetany, and have been referred to as "fits" of tetany. They may be recognised by the clinical signs of generalised increased muscle tone. Such signs are seen in the intoxications due to strychnine and tetanus, in the metabolic disorders associated with parathyroid insufficiency and with the early transient stages of hypocalcaemic states, and in certain neuromuscular disorders such as "Scotch cramp."

Section 3 includes drug intoxications in which one gets maniacal behaviour with or without motor seizures, the latter occurring when the dosage of the drug is very high. The best example of these is fluoroacetate intoxication (Chenoweth & Gilman, 1946), but similar signs may develop with benzedrine.

Section 4 comprises the anxiety states not associated with the the canine-hysterias, *i.e.*, those accompanying naturally occurring neuroses and the neurasthenias produced by experimental techniques (Babkin, 1938; Anderson, 1939). Finally in section 5 we have a group of miscellaneous disorders in which motor seizures occur inconstantly but occasionally. These include the cases of renal insufficiency in which so-called "uraemic convulsions" occur, the occasional case of an eclampsia-like syndrome in pregnant bitches with "convulsions," and the "convulsions" seen in extreme hypo-glycaemia (Mann & Magrath, 1922), but which are by no means as constant in the dog (Drabkin & Ravdin, 1937) as they are in the rabbit.

Conclusions

The wide range of canine diseases in which occur clinical signs of nervous dysfunction of an epileptic character makes the investigation of this field of veterinary medicine one of special interest and peculiar difficulty. However, the successful production of one aetiological entity under experimental conditions, namely canine-hysteria, means that we can now define the clinical and pathological features of one syndrome with some accuracy, knowing that no other complicating factors are present. We can use this information as a yardstick with which to attempt the clinical differentiation of other naturally occurring syndromes and in due course define the conditions necessary for their experimental production.

The classifications and diagnostic criteria proposed in this paper have been put forward in a tentative way only, in the hope that they may serve a useful purpose as a basis for further work and discussion on this group of canine diseases.

Acknowledgment.—During the preparation of this paper, discussion with Mr. J. D. Steel, of the University of Sydney, has been most helpful.

REFERENCES

ALAJOUANINE, T. (1947.) *Proc. roy. Soc. Med.* **40.** 686.
ALLEN, F. F. (1945.) *Amer. J. Psychiat.* **102.** 67.
ANDERSON, O. D. (1939.) *Amer. J. Physiol.* **126.** 421.
ANON. (1735.) The Sportman's Dictionary or the Country Gentleman's Companion. 2 vols. Vol. I. London: Hitch, Davis and Austen.
BABKIN, B. P. (1938.) *Edin. med. J.* **45.** 615.
CHENOWETH, M. B., & GILMAN, A. (1946.) *J. Pharm. exp. Ther.* **87.** 90.
DRABKIN, D. L., & RAVDIN, J. S. (1937.) *Amer. J. Physiol.* **118.** 174.
FOERSTER, O., & PENFIELD, W. (1930.) *Brain.* **53.** 99.
HALL, C. S. (1947.) *J. Hered.* **38.** 3.
HILL, J. WOODROFFE. (1892.) The Management and Disease of the Dog. 4th ed.; pp. xii + 437. London: Swan, Sonnenschein.
HURST, E. W., COOKE, B. T., & MELVIN, P. (1943.) *Aust. J. exp. Biol. med. Sci.* **21.** 115.
JONES, J. H., Schmidt, H., *et al.*, (1943.) *Texas agr. exp. Sta. Bull.*, **630.**
KELLAWAY, C. H., TRETHEVIE, E. R., & TURNER, A. W. (1941.) *Ibid.* **18.** 225.
MACINTYRE, A. B., TREVAN, D., & MONTGOMERIE, R. S. (1948.) *Vet. Rec.* **60.** 12.
MADSEN, L. L. (1942.) U.S.D.A. Yearbook. "Keeping Livestock Healthy"; pp. 658. Washington: Government Printing Office.
MANN, F. C., & MAGRATH, T. B. (1922.) *Arch. int. Med.* **30** 171.
MEGNIN, —. (1881.) *Bull. Soc. cent. Méd. vét., Paris* (1882.) **11.** 380.
MOORE, L. A., & SYKES, J. F. (1941.) *Amer. J. Physiol.* **134.** 436.
NEWELL, G. W., ERICKSON, T. C., GILSON, W. E., & ELVEHJEM, C. A. (1947.) *Proc. Soc. exp. Biol. Med.* **65.** 115.
NOCARD, —. (1882.) *Bull. Soc. cent. Méd. vét., Paris.* **11.** 375.
PARRY, H. B. (1941.) Unpublished records. Sydney Univ. Vet. Sch.
———. (1942.) *Ibid.*
———. (1948a.) *Lancet.* **1.** 32.
———. (1948b.) *Vet. Rec.* **60.** 389.
PERDRAU, J. R., & PUGH, L. P. (1931.) *Jour. Path. Bact.* **33.** 70.
PENFIELD, W., & ERICKSON, T. C. (1941.) Epilepsy and Cerebral Localisation. Springfield, Ill.: C. C. Thomas.
STEEL, J. D., & WYKE, B. D. (1947.) *Aust. vet. J.* (Oct.)
———. (1948.) Unpublished record. Sydney Univ. Vet. Sch.
TAYLOR, J. (1931.) Selected writings of Dr. Hughlings Jackson.
WILSON, S. A. K. (1940.) Neurology. London: Arnold. Vol. II p. 1471.

Discussion

Mr. E. O. Longley referred to Mellanby's film, saying that he felt sure that the syndrome it represented was not identical with the clinical condition so well remembered in veterinary practice. The latter, in his experience, never presented the picture of a progressive degenerative nervous disease with, for example, persistent ataxia, as Mellanby's dogs showed. Nevertheless he thought it did not do to disallow the agene theory as some seemed inclined to do. No doubt there was a correlation, but the story was incomplete. It was perhaps of interest that he had the assurance of high medical authorities that epilepsy in man and no doubt also the anxiety neurosis was on the increase in this country. He had heard the possible role of agenised flour discussed as a cause.

Mr. Longley went on to say that the trigger-mechanism of the clinical hysteria in the dog remained strangely obscure. Reference to the acousticogenic fit so readily induced experimentally in certain rodents might be profitable, bearing in mind also that the dog had audio-sensitivity well above the sound-frequency range in man. It might thus be the case that the fits were triggered off in susceptible dogs in a manner similar to the seizures in rodents, but by sound stimuli which, being higher in frequency than the human ear could detect, were not sensed by human observers. This was only a suggestion, but it would be a matter of no difficulty to examine it experimentally.

He then suggested to Mr. Parry that if his dogs were showing generalised discharges with the epileptiform character of the electroencephalograms which they had been shown, it was not only entirely certain that the dogs were unconscious, but that they would also be incapable of the comparatively well co-ordinated motor activity of the hysterical dog. He asked him whether he had had recourse to the well-known epileptogenic procedures involving hyperventilation, alkalaemia, and hypoglycaemia, on his agenised dogs? These could be very useful experimentally, and it would be most interesting to know whether they would be operative in this case, and if so what pattern of seizure would ensue?

Major Hamilton Kirk wanted to know if all the cases or a proportion only were due to agenised wheat gluten. It would be presumptuous on his part to dispute the conclusions arrived at by some workers who sought to prove that hysteria was caused by agenised flour, but they did not entirely hold water, as 90 per cent. of British flour was agenised; and as 90 per cent. of baker's bread was made with agenised flour he would expect to find most of the dog population affected with hysteria. He had not seen a case for two years and dog biscuits were still being made with agenised flour. He had had a letter from Switzerland saying that the bread there was never made from bleached or agenised flour, but before the war and since 1945 dogs had been fed on Spratt's biscuits made in England and yet there was no hysteria in Switzerland although the dogs had access to English dog biscuits and to agenised flour from the U.S.A. He had had a letter from the small-animals clinic of the Brussels veterinary college which said that at present no hysteria existed. There was no adulteration of the native flour but foreign flour was imported and it was necessary to look elsewhere for the cause of the disease. From a French veterinary college he had a similar letter and all three seemed to prove, and in fact stated, that agenised flour could not be the cause of hysteria. He agreed that the symptoms as described by Mellanby were not hysteria as the veterinarian saw it in the field. He did not think epileptic convulsions were a prominent part of hysteria; the most prominent was the extreme hyperaesthesia and dashing about in full consciousness. They knew that if they changed the diet the hysteria stopped; they could change it from one brand or make of bread or biscuit to another brand but they still changed to wheat gluten. His own idea was that the hysteria arose from an autotoxaemia arising from some deleterious but still undetected substance in the individual animal's bowel. A small amount of poisonous fungous growth on wheat might be contained in one sack of flour and if they fed dogs on another sack of flour in which there was no poison animals would not have hysteria. This seemed to point to the fact that wheat gluten had little to do with the causation of hysteria. He could not remember when he last had a case of hysteria, although he had permanent charge of a kennel of 300 greyhounds the food of which contained a large proportion of wheat gluten.

Dr. Parry: He did not consider that agenised flour was the only cause of dietary canine hysteria; there were at least five other syndromes in which there was no question of infection or parasites being involved. In canine hysteria there were no focal neurological signs and the clinical signs disappeared when the dietary régime was corrected. He thought canine hysteria comprised a number of clinical types: there was the syndrome of epileptic "grand-mal" seizure as well as the more usual "running barking fit." These had been produced at will—ten or 15 seizures every day for a week—until he changed the diet and replaced the wheat gluten by casein, the wheat gluten being prepared from agenised flour. In France they did not have agenised flour, but from time to time they imported American flour. In 1944 they had an epizootic of canine hysteria coinciding with the arrival of the American troops on the Riviera Beach-head; a large proportion of the dogs in the province were affected but they all recovered and the epizootic disappeared in three months. The previous epizootic was in central France in 1930. How did they explain observations of that kind?

Major Kirk: In France the flour was not subjected to bleaching or extraction under chemicals, and yet there were cases of hysteria. The work of Sir Edward Mellanby did not explain all cases of hysteria; the cases of French hysteria were quite different.

Dr. Parry: From the published descriptions the French form of the disease appeared to be identical with our canine hysteria. With regard to Mellanby's work, a cardinal point was that the clinical response was very closely related to the amount of agenised protein ingested. Dogs eating 5 grammes of wheat gluten per kg. of body weight per day showed no sign of hysteria; at 10 to 12 gramme levels the dogs showed anxiety and became hysteria-prone; and at 20 grammes of wheat gluten daily the dogs showed "spontaneous" running and "grand-mal" seizures. In clinical practice they were unlikely to reach that level. The quantitative factor helped to explain why in clinical practice they had with certain batches of biscuits a series of cases; the biscuit was sufficiently hysteriagenic to raise the animal from the sub-hysteria-prone level to the hysteria-prone level when clinical signs occurred. There were other factors apart from agenised flour. Hysteria-prone level was affected by other constituents in a diet, *e.g.*, the number of calories derived from fat. If this was increased the liability to hysteria was reduced. Water retention was another factor; if the dog was in a hysteria-prone state large quantities of water by the stomach precipitated the motor episodes; if they reduced water retention, they reduced the liability to motor episodes.

Dr. J. T. Edwards: The original experiments were with the feeding of dogs on a composite diet and the control was casein.

Dr. Parry: The diets were made up of highly purified constituents and the only difference between the hysteriagenic and control diets was that the former contained wheat gluten and the latter casein in iso-caloric amounts. The wheat gluten was bought from a commercial milling company and it was prepared from agenised flour.

Dr. Edwards: In the Mellanby experiments the dogs were fed on wheat gluten not agenised and the attacks were precipitated when the wheat incorporated in the diet was agenised. He thought Dr. Parry's announcement was that the dogs' hysteria was precipitated by the wheat gluten alone (untreated) and cured by inserting another protein, namely, casein.

Dr. Parry: There was no data on wheat gluten definitely not prepared from agenised flour. With Mellanby's experimental technique it was not possible to be sure that control animals were eating enough wheat gluten to exceed the maximum tolerated daily dose.

Dr. Edwards: He had studied Mellanby's work on vitamin A, where he claimed to produce that sort of thing by administering vitamin A deficiency diets or cereals. Mellanby might have been wrong there. He wished Mellanby had been present, as his work had been exposed to criticism. His experiments must prove it was the agenisation, otherwise Major Kirk's point might arise, that they might get the condition when dogs were fed on flour not agenised and not bleached.

Major R. F. Wall: Clinical observations bore out the theories of the hysteria level. In the U.S.A., when rabies was prevalent, many dogs in fits were shot, but some escaped and when they returned home they were perfectly normal. The thing that struck him was that nearly all the dogs with fits were house-fed dogs, but with the working (hunting) dogs he did not remember seeing one with hysteria. In the Southern States they were largely fed on maize meal and hysteria was a rarity; it did occur but he did not remember more than six dogs so affected. He could not discuss the academic aspect, but it seemed to him that there must be some factor besides agenised flour. He had a kennel with 220 dogs fed on meat and rusk from a certain firm. The time came when half a dozen had hysteria, then the 200 developed it. The rusk was stopped and no more cases of hysteria occurred. He tried rusks again and one dog had hysteria after one feed, and so it went on—through the whole lot. Certain dogs manifested hysteria after one to three feeds from 24 hours to four days. In a kennel of 62 or 63 greyhounds all had hysteria and he put them on sedatives. To his personal knowledge those dogs were fed on wholemeal bread from flour grown, milled and baked locally. They had hysteria badly. The same bread was rebaked and fed to the dogs, none of which developed hysteria. Like Major Kirk, he had not had a case of hysteria for more than two years until

the previous week, when a bitch developed hysteria. Since then he had met with a number of cases of hysteria and all the dogs had been fed on horse meat, stale bread, milk and vegetables.

In reply to Mr. WRIGHT, who asked if the experimental dogs returned to normal lives after they had gone through the experimental process, Mr. Parry said that the majority did so. Five or six hysterical assays were carried out on most of the animals; one or two of the younger dogs used when ten to 12 weeks old became blind temporarily. One puppy, which had a very large amount of wheat gluten, was completely blind at the end of the experiments. The cause was not known. This was the only residual neurological defect observed in any of the dogs.

Mr. W. A. POOL: Were the brain lesions in the hysteria-prone dogs visible to the naked eye?

Mr. PARRY: There were no macroscopic lesions and nobody had yet been able to find any microscopic lesions. With other epileptic states, particularly following virus infection, small lesions were formed due to the encephalitis, which might possibly act as epileptogenic foci. He fancied a virus encephalitis or an infective encephalitis produced fits in two ways; either by the immediate effect of the infection on the whole nervous system or later some three to four weeks after the initial febrile septicaemia, when small post-encephalitic residua became epileptogenic and began to set up dysrhythmia.

Miss STAINTON mentioned the case of a dog which had distemper in the autumn of 1947, made a good recovery and was apparently normal, but now was going blind.

Professor J. McCUNN: Did cats ever show signs of hysteria?

Dr. PARRY: He had never seen dietary hysteria in cats, but there was evidence in the literature that cats could suffer from a hysteria-like disorder. The work on agene intoxication had shown that different species varied a great deal in their susceptibility. It was impossible to cause intoxication in the chicken, guinea-pig, rat and mouse; the cat required very large amounts of agenised flour before showing convulsions, while the monkey showed no clinical signs, but there were changes in brain rhythm. The dog was peculiarly prone to agene intoxication while there was no evidence yet that man was affected.

Miss UVAROV said she was glad to hear Dr. Parry describe epilepsy as a "syndrome," with its differentiation into three stages, *i.e.*, pre-hysteric, hysteric and post-hysteric. These stages were most important to the clinician, who on encountering a patient with signs of the disease of the nervous system, had (1) to consider whether the diagnosis was epilepsy—or any other condition affecting the nervous system; (2) search for the cause; (3) treat the cause and (4) if present—and this was most essential to the owner—treat the fits.

She had hoped that there would have been more contributions, in the discussion, on the clinical aspect, and that some of the practitioners would have shared their views on the numerous sedatives and narcotics now in use.

Clinically, she subdivided "Idiopathic epilepsy" into the following groups:—

(1) *Original type*—sudden onset. Dog starts barking, running wildly; shows no signs of fear.

(2) *Neurasthenic type*—fear is the predominant feature; sensibility is not lost; responds to psychological treatment.

(3) *Mental type*—dog completely wild, often vicious. No recognition, almost appearing like complete madness.

Miss Uvarov asked Dr. Parry if any predisposing causes, *e.g.*, age, breed, environment, or long fasting, had been observed by him to be of any significance.

What could be the explanation of the fact that some people could never keep a dog without it becoming stubborn, "nervous," and later completely bad tempered? It had been noticed that most such owners were themselves of the nervous type.

Did Dr. Parry recognise the effect of auricular acari in hounds—which appeared to be the oldest form of canine hysteria as observed by the French workers as early as 1836?

Could Dr. Parry explain the state of hysteria as occurred in phantom pregnancy in the bitch?

She associated herself with Dr. Parry in that the film shown by Sir Edward Mellanby did not show canine hysteria as seen in practice.

Dr. PARRY: Auricular acariasis was first described by Mégnin and Nocard in 1881-82 as the cause of a syndrome was should now call canine hysteria. Phantom pregnancy was an anxiety state in the bitch at the end of the lactation and he did not know what the cause was. During phantom pregnancy there were almost certainly profound changes in the distribution of body fluid and he suggested this might be a factor. Nephritis with renal insufficiency led to fluid retention again. He thought that eliminating foreign bodies could act as a precipitating cause of hysterical episodes. He believed younger animals to be more prone.

On the nervous disorders among Arctic dogs, many of the Canadian reports were extremely confused and dealt with at least four different syndromes. He could not subscribe to the view that there were necessarily or invariably morphological organic lesions in pathological disorders. Many diseases were due to a functional insufficiency of an organ caused by biochemical disturbances, which were not associated with any organic lesion, *i.e.*, the lesion was "biochemical." Furthermore, if organic lesions were demonstrated it was essential to show that they caused a functional deficit of a magnitude sufficient to account for the clinical observations.

CLINICAL COMMUNICATION

Tuberculosis of the Membrana Nictitans in a Heifer

T. A. M. CHAMBERS, M.R.C.V.S.

WOLVERHAMPTON

Subject.—Eighteen-month-old Freisian heifer.

History.—The heifer had had a discharging eye for two months, also a "lump" under the ear.

Examination.—There was a profuse mucopurulent discharge from the eye, originating from an enlarged granulomatous third eyelid. The "lump" was a greatly enlarged subparotid gland.

Diagnosis.—Infected tumour of third eyelid with glandular involvement due either to metastases or infection.

Treatment.—The third eyelid was removed under cocaine anaesthesia and was found to be granulomatous with purulent nodules.

Pathological Report.—Smears showed the presence of acid-fast bacilli with the characters of *M. tuberculosis.*

Histological examination of paraffin sections confirmed the diagnosis of tuberculosis. Numerous tuberculous nodules were present throughout the stroma of the eyelid.

Remarks.—This was a case of primary tuberculosis of the membrana nictitans with spread to the subparotid gland. The source of infection is unknown, but most probably it was due to sputum from another cow being coughed into the eye.

Acknowledgment.—The pathological examination was carried out by Mr. J. S. Beattie, of the Royal (Dick) Veterinary College Pathological Department.

ABSTRACTS

[Transmission and Immunisation Studies on Equine Influenza. JONES, T. C., GLEISER, C. A., MAURER, F. D., HALE, M. W., & ROBY, T. O. (1948.) *Amer. J. vet. Res.* **9**, 243.]

Experimental studies on equine influenza during the period 1939-47 inclusive are described. The object of the work was to develop means of prevention of morbidity and mortality due to the disease and its complications. A detailed description of materials and methods is given. The usual laboratory animals, and calves, cats and swine, were found to be unsuitable for use in the study of the equine influenza virus and it was necessary to maintain the virus by serial passage in young horses. With one strain of virus this technique was alternated with the storage of tissues in a dry ice cabinet at —70° C., the virus being maintained thus since 1941. The virus could be preserved in equine tissues at —70° C. for 20½ months, and also survived desiccation and subsequent storage at 21° C. or 4° C. for 119 days. It was shown in colts that convalescent or immune serum from horses would neutralise the virus. Attempts to cultivate the virus in developing chick embryos and in tissue cultures were unsuccessful.

The symptoms and lesions of the experimentally induced disease, which are fully described, closely resembled those of the uncomplicated naturally acquired mild form. The virus could be demonstrated in blood, serum, nasal washings and nasal or turbinate mucosa. The spleen was to be preferred as the source of virus during the early febrile stage of the disease. Parenteral injection of infective spleen suspensions of a virus of low virulence produced a mild febrile reaction followed by immunity to further inoculations. This vaccine showed promise in immunising against natural infection, and a desiccated virus vaccine which might prove of value was also prepared. The methods of preparation of the two vaccines are described in detail.

C. A. E. B.

[Treatment of *Streptococcus agalactiae* Mastitis by Single Injections of Penicillin in Retaining Vehicles. Stults, A. W., & Foley, E. J. (1948.) *J. Amer. vet. med. Ass.* **113.** 68-72. (7 tables.)]

The experiment involved 500 to 600 milking cows, and aimed at treating all those infected with *Str. agalactiae*. All the animals were machine milked at 12-hourly intervals. No attempt at segregation was made.

The retaining vehicles used have been described by the author elsewhere. They are essentially mineral oil and water emulsified with lanolin derivatives. Infected quarters were treated with a single injection of 100,000 units of crystalline sodium penicillin in one of two types of vehicle. Routine 12-hourly milkings were continued after treatment. Samples were drawn at ten- and 21-day intervals following treatment, and at monthly intervals thereafter.

When aqueous solutions are employed, best results follow repeated injections of 20 to 50,000 units. Of 395 quarters treated with single injections of 100,000 units in retaining vehicle, 342 (or 86·5 per cent.) were cured. Of the 45 injected a second time 41 (or 91 per cent.) were cured.

When 40 mg. of tyrothricin was included in the injection, a marked inflammation of the udder occurred 12 hours after the injection, and the results were not as good as when penicillin alone was used.

The water-in-oil vehicles increase the efficiency of the penicillin following a single injection, by greater dispersion into the upper portion of the duct system of the udder, and gradual release of the drug occurs, at therapeutic levels in the milk.

P. K.

* * * * *

[Production and Use of New Biological Products against Swine Erysipelas in Poland. Kaplan, M. M. (1948.) *Vet. Med.* **43.** 174-179 (11 refs.).]

This article gives details of the preparation and application in Poland of (*a*) Staub's attenuated viable vaccine and (*b*) Muromcev's formalin-killed vaccine. Both have received field trials and appear to be safe and to give protection for about four months. The former is regarded as the more practicable—it is easier to prepare and one injection only is required as against two or three injections of the latter. A mass trial of Staub's vaccine under field conditions has been planned for 1948.

A method of producing immune serum in horses is described. The principle of the method is the subcutaneous injection of agglutinated organisms in place of the intravenous injection of large volumes of culture. This is claimed to produce high quality sera without causing the well-known toxic symptoms in the horses.

A. W. G.

REVIEW

[The Epidemiology of Parasitic Diseases with Special Reference to Studies with Nematode Parasites of Sheep. By H. McL. Gordon. (*Aust. vet. J.* **24.** 2.)]

The entire second number of the *Australian Veterinary Journal*, volume 24, is devoted to an extended article by Dr. Gordon on the epidemiology of parasitic diseases, with special reference to the nematode parasites in sheep. The many facets of helminth infestation and its control are examined. The significance of the infection and contamination phases, and their association with the parasite, diet, animal husbandry, climate, and anthelmintic treatment are extensively reviewed and illustrated. Dr. Gordon is to be congratulated on a brilliant review of a major problem of the sheep industry.

In order that manufacturers, distributors and all interested in food science can keep abreast of the latest advances in knowledge, a new journal, "Food Science Abstracts," is to be published for the Department of Scientific and Industrial Research in the next year. Abstracts will deal with meat (including pig flesh), poultry, game, fish, dairy produce, fats, fruit, vegetables and grains, and will be published on alternate months at 6s. per volume.

* * * * *

Weekly Wisdom

The American people have decided that poverty is just as wasteful and just as unnecessary as preventable disease.—President Truman

Victoria Veterinary Benevolent Fund

MEETING OF COUNCIL

A meeting of the Council of the Victoria Veterinary Benevolent Fund was held at No. 10, Red Lion Square, London, W.C.1, on Friday, November 26th, 1948, when the following members were present:—

Mr. Arnold Spicer (President) in the chair, Professor J. B. Buxton, Mrs. M. Buxton (President, Ladies' Guild), Major J. J. Dunlop, Mrs. D. I. Glover, Professor J. McCunn, Lt.-Colonel P. J. Simpson, Messrs. H. P. Standley, H. W. Steele-Bodger, Mrs. E. B. Wooldridge (Secretary, Ladies' Guild), Dr. W. R. Wooldridge. Mr. C. W. Francis, Accountant, was in attendance.

Minutes.—The minutes of the previous meeting, having been published, were taken as read and signed as correct.

Apologies for Absence.—Letters of apology for absence were received from Mr. R. E. Glover, Captain W. R. McKinna, Captain J. W. Procter, Colonel G. B. Rees-Mogg, Captain A. Whicher and Professor G. H. Wooldridge.

Correspondence.—Letters were read from:—

(*a*) Captain A. Whicher, thanking the Council for his election to Council.

(*b*) The Secretary, Royal College of Veterinary Surgeons, stating that Mr. R. E. Glover and Captain T. M. Mitchell had been elected as representatives on the Council of the Fund.

(*c*) The Australian Veterinary Association, stating that the New South Wales Division acted as forwarding agents for the A.V.A. and that all Divisions of the A.V.A. took part in providing the food parcels so kindly sent to our recipients. Also that the letter of thanks from the Fund sent to the New South Wales Division had been circulated to all the Divisions of the A.V.A.

Secretary's Report.—The Hon. Secretary reported on his visit to Brighton and his interview with case 215a. His action taken in consultation with the Executive Committee was approved.

He further reported on his visit to Aberystwyth to attend the meeting of the South Wales Division of the Ladies' Guild, which was well attended and enthusiastic, and in connection with this read a letter from the President of the Division stating that a North Wales Division had been formed.

Treasurer's Report.—The Treasurer, Professor J. B. Buxton, reported:—

(i) That on August 19th, 1948, he had arranged for the investment of the balance standing to the credit of the Donations Account in the purchase of £394 9s. 3d. 2½ per cent. Funding Stock.

(ii) That the sum of £211 2s. 5d. Hunting Fund Account balance for 1947, as disclosed in the balance sheet, had been invested in the purchase of £210 5s. 8d. British Transport Guaranteed Stock, 3 per cent., 1978-88, in the name of the Victoria Veterinary Benevolent Fund.

The action of the Treasurer was approved.

Accountant's Report

The Accountant submitted the following report of new subscriptions and donations received since the previous meeting:—

Subscriptions	£	s.	d.
Bexhill Horse Show	20	0	0
East Berks. Horse Show Society (per Col. Simpson)	11	15	0
F. B. Greer	0	10	6
W. Nairn	3	0	0
V.V.B. Fund Ladies' Guild	300	0	0
	£335	5	6

Donations	£	s.	d.	£	s.	d.
Collecting Boxes						
A. Whicher	4	6	2			
Central Veterinary Society	5	6	6			
Derbyshire Veterinary Association	3	7	0			
Eastern Counties Veterinary Society	4	14	0			
J. T. Morton	0	12	6			
Scottish Metropolitan Division	8	17	8			
Sussex Division—N.V.M.A.	8	8	6			
				35	12	4
Anonymous—"In Memoriam"				100	0	0
Association of State Veterinary Officers				5	5	0
Cleobury Mortimer and Ludlow Show (per Capt. Yarrow)				1	1	0
Dumfries and Galloway Division				5	5	0
Eastern Counties Veterinary Society Shipley Bowls				9	12	6
Estate of Ellen Clanchy—Bequest	100	0	0			
Interest accrued	1	4	3			
				101	4	3

	£	s.	d.
Ladies of North of England V.M.A.	7	0	0
Lincolnshire and District V.M.A.	5	0	0
National Greyhound Racing Club	5	0	0
D. S. Murray	0	15	0
J. J. O'Donovan	2	2	0
R.V.C. Academic Staff—"In Memory of Miss Barber"	3	0	0
Scottish Metropolitan Division	10	0	0
Arnold Spicer	100	0	0
Students Union Society, Streatley, Proceeds of Sale of Bicycle	3	12	6
Sutton and Cook	2	2	0
Sussex Division N.V.M.A.	10	0	0
	£406	11	7

Christmas Fund	£	s.	d.
Ladies' Guild	100	0	0
H. Stainton (Crufts Show)	26	5	0
	£126	5	0

It was resolved, "That the sincere thanks of the Council be conveyed to the respective donors, and especially to the Ladies' Guild, Mr. Arnold Spicer and to the Anonymous donor, for their very generous subscriptions and donations."

CASES

259. Widow; age 76. The Hon. Secretary reported the death of this recipient which took place on October 3rd, 1948.

249. The Hon. Secretary reported that on the instructions given at the previous meeting, he had written stating the reasons for discontinuing this recipient's grant, and read her reply of appreciation for the assistance which had been given to her.

52. Widow; age 70. A report from the Executive Committee, that they had increased her grant from £1 to £2 per week as from June 1st, in view of her altered circumstances, was presented. The action of the Executive Committee was approved.

256. Widow; age 89. This case was further reviewed in consequence of correspondence received since the last meeting, and having regard to a report of the Executive Committee.

It was resolved, "That the Hon. Secretary be asked to make a personal visit to this recipient and report to the Executive Committee, who shall take such action as may be deemed necessary."

215a. Daughter of late member; age 83. The Hon. Secretary intimated that in accordance with the decision arrived at at the previous meeting he had visited this case. The Executive Committee, after being informed of the circumstances of this recipient, decided that the grant should be transferred to her sister (215b) and increased from 10s. to £1 per week. The action taken by the Executive Committee was approved and it was agreed that the grant be continued.

263. M.R.C.V.S.; age 23. A letter applying for assistance on account of illness was read, but as the application form had not yet been returned, it was agreed that the case be left in the hands of the Executive Committee.

262. Widow; age 50. At present in employment. Has one daughter; age 20. After careful consideration of this case it was decided that the present circumstances of this applicant do not appear to justify immediate financial assistance, but in conveying this decision the Hon. Secretary should make it known that her case will be further considered in the event of her circumstances changing and a further application being submitted.

Despite the importance of putting a bigger acreage under the plough, the real emphasis of our farming economy must fall upon livestock. Stockbreeding is the traditional pivot of British husbandry, and we know how to do it as well as any nation in the world. It is not for nothing that almost all the best breeds of cattle, sheep, pigs (and horses) have English names or that stud farms the world over look to us for their foundation stock.—Mr. Tom Williams (Minister of Agriculture) speaking at Preston.

* * * * *

The University of Missouri has announced plans for a meat laboratory; a large building where slaughtering, curing, smoking, cutting, canning, quick freezing, and all manner of meat processing and cooking, will be studied with a view to developing improved processes.—*Vet. Med.*

NOTES AND NEWS

Diary of Events

Jan. 19th.—Meeting of the West of Scotland Division, N.V.M.A., at Glasgow (Central Station Hotel), 2.30 p.m.
Jan. 21st.—Annual Meeting of the Mid-West Division, N.V.M.A., at Bristol (Berkeley Café, Clifton), 2 p.m.
Jan. 21st.—157th Annual Ball of R.V.C., at Grosvenor House.
Jan. 21st.—Meeting of the Midland Counties Division, N.V.M.A., at Birmingham (Grand Hotel), 2.30 p.m.
Jan. 21st.—Meeting of the Lincolnshire and District Division, N.V.M.A., at Grantham (George Hotel), 2.15 p.m.
Jan. 22nd.—Meeting of the Biochemical Society, at the British Postgraduate Medical School, Ducane Road, Hammersmith, W.12, 11 a.m.
Jan. 25th.—Meeting of the Derbyshire Division, N.V.M.A., at Sutton Bonington (Midland Agricultural College), 2.15 p.m.
Jan. 27th.—Meeting of the Western Counties Division, N.V.M.A., at Exeter (Royal Clarence Hotel).
Jan. 28th.—Annual Meeting of the Yorkshire Division, N.V.M.A., at Leeds (Hotel Metropole), 2.30 p.m.
Feb. 8th.—Scientific Meeting of the Zoological Society of London, Regent's Park, N.W.8, 5 p.m.
Feb. 18th.—Meeting of the Sussex Division, N.V.M.A., at Brighton (Old Ship Hotel), 2.30 p.m.
Aug. 8th to 13th.—Fourteenth International Veterinary Congress, Central Hall, Westminster, and Church House, Westminster, London, S.W.1.

* * * *

N.V.M.A. Membership Subscription

The attention of members is drawn to the following Article of Association of the N.V.M.A.:—

> 35. During the first week of March in each year, notice shall be sent to all members whose subscriptions shall be unpaid. Should the subscription of any member remain unpaid by the 30th day of June, he shall cease to be entitled to receive the publications of the Association or to attend or vote at any General Meeting but shall remain legally liable for his subscription. If the subscription of a member shall be in arrear for one year he shall cease to be a member of the Association.

In the past, through failure to pay their subscriptions when due, the Association has been in the unfortunate position of having to remove the names of a number of members when it had been only a matter of inadvertence that the subscription had not been paid. The Officers of the Association will be pleased if this position can be avoided in future by members recognising the need to have their subscriptions paid in good time.

The majority of members find a banker's order the most convenient method of payment, and the General Secretary will be glad to send one of these forms to any member desirous of adopting this system.

* * * * *

PERSONAL

ILLNESS OF SIR FRANK WARE

Our readers will learn with much regret that Sir Frank Ware, C.I.E., F.R.C.V.S., formerly Director of Animal Husbandry to the Government of India, entered the Tropical Diseases Hospital, 23, Devonshire Place, W., on January 6th, for the treatment of sleeping sickness. It will be recalled that Sir Frank led the mission appointed by the Secretary of State to visit West Africa to investigate the livestock industry in the Cameroons under British mandate, with particular reference to meat production. He has been unwell since his return early in December and last week it was confirmed that he is infected with Trypanosomes. All members of the profession and his many other friends will hope most earnestly for the early and complete success of the treatment he is undergoing.

* * * * *

Birth.—Oxley.—At The Ordnance Nursing Home, Southampton, on January 4th, 1949, to Evelyn, wife of H. Oxley, M.R.C.V.S., 18a, Spring Crescent, Southampton, a daughter.

Marriage.—Watson—Ball.—At St. Patrick's, Nuthall, Nottingham, on January 1st, 1949, Capt. A. H. Watson, M.R.C.V.S., only son of Mr. T. K. Watson, Edinburgh, to Eileen, youngest daughter of Mr. and Mrs. G. Ball, Nuthall, Nottingham.

* * * * *

R.C.V.S. OBITUARY

Hally, James Charles, George Street, Wellington, Somerset. Graduated London, December 17th, 1902. Died January 7th, 1949.

Roach, William, F.R.C.V.S., York House, York Road, Exeter. Graduated London, July 3rd, 1879; Fellowship, July 28th, 1891. Died January 3rd, 1949; aged 91 years.

Thompson, Arthur Joseph, Lt.-Colonel R.A.V.C. (retd.), of Auckland, New Zealand. Graduated London, December 17th, 1902. Died (suddenly) July 3rd, 1948.

Major Alex. Douglas

A Further Tribute

The death of Major Alex. Douglas removes one of our members who, in spite of frequent attacks of illness, played a most important part in the field of veterinary public health. He was one of the first to urge a determined effort to eradicate tuberculosis from the dairy herds of the country and his labours helped to build up the outstanding position of the cattle of Ayrshire to-day. From this work the breeders of Ayrshire cattle have reaped a rich reward, but it was only a year ago that they made a presentation to Major Douglas as tangible evidence of their appreciation.

He qualified in 1905 at the London College; later at Manchester he obtained his Diploma in Veterinary State Medicine, and returned to his native town of Kilmarnock to assist his father in mixed practice.

About 1913, when Ayrshire County Council decided to appoint a whole-time Veterinary Inspector, he was the successful candidate and early commenced to put his ideals into practice. War service interrupted this work, but afterwards he again took up the task with renewed energy and initiative.

To achieve his aims he built up a very strong veterinary department. In the late twenties it happened that Major J. Gilray McGregor addressed the West of Scotland Division on a Scheme for the Eradication of Bovine Tuberculosis. This was later brought before the Scottish Branch and remitted to a committee for consideration and for amendment if necessary. In this work Douglas was enthusiastic and his contribution was outstanding. The Scottish Branch forwarded their scheme to the N.V.M.A. and without amendment it was adopted by the parent body.

The high quality of Douglas's work became well known throughout the profession and it was no surprise that when the Ministry of Agriculture took over the work of improving the health of the dairy cow Douglas and other pioneers were called to Headquarters in London.

After the lamented death of Capt. Robert Simpson he was appointed Deputy Chief of the Animal Health Division. The task of organising this new department would have been a Herculean one to a man of robust health, but may have been too much for one like Douglas, whose physical strength was not equal to his great nervous energy, and so after seven years we learned with regret he had retired after a long period of invalidity. He then went to the Perthshire town of Blairgowrie, where he lived very quietly, enjoying as far as he was able the pleasures of his garden.

The strain and worry of organising the big new department controlling the production of milk seemed to play a large part in undermining his health and hastened the final phase, so that to-day we mourn the loss of a fellow member whose services honoured our profession and will long provide a splendid memorial to his devoted life-work.

W. N.

* * * * *

FURTHER AMENDMENT OF POISONS LIST AND RULES

The Poisons Board has made certain recommendations to the Secretary of State for the further amendment of the Poisons List and of the Rules made under Section 23 of the Pharmacy and Poisons Act, and he therefore proposes to make Statutory Instruments which will effect changes in the law relating to poisons. By the operation of one of these, registered veterinary practitioners will be given the same facilities for obtaining, supplying and dispensing poisons as are enjoyed by registered veterinary surgeons.

The remainder are as follows:—

(1) 6-morpholino-4: 4-diphenylheptane-3-one and its salts will be added to Part I of the Poisons List.

(2) Dinitrocresols will be taken out of Part I and added to Part II of the Poisons List.

(3) Bis-bis-dimethylaminophosphonous anhydride, diethyl-paranitrophenyl thiophosphate, hexaethyl tetrophosphate and tetraethyl pyrophosphate will be added to Part II of the Poisons List.

(4) 6-morpholino-4: 4-diphenylheptane-3-one, will be included in the First and Fourth Schedules to the Poisons Rules.

(5) Dinitrocresols will be exempt from the restrictions which apply to First and Fourth Schedule poisons when sold in the form of agricultural and horticultural insecticides or fungicides. Such preparations will be excluded from the general exemption from control of all substances containing dinitrocresols and not being preparations for the treatment of human ailments; they will be the only preparations containing dinitrocresols and subject to the control of the poisons law which may be sold by listed sellers of poisons.

(6) The four phosphorous compounds already named will be exempted from control when sold in any form other than agricultural or horticultural insecticides or fungicides. These preparations, and the similar preparations containing dinitrocresols, will be subject only to the restrictions imposed by their being included in Part II of the Poisons List and in the Seventh Schedule to the Poisons Rules: the form of words with which they are to be labelled will draw attention to the danger of using the preparation in an enclosed place and of allowing it to come into contact with the skin.

(8) Rule 18 will be amended to provide that a poison may be labelled with the "Approved Name" given it by the General Medical Council.

(9) Rule 30 will be extended to include insulin in the list of poisons which may be manufactured under the supervision of a duly qualified medical practitioner.

(10) Rule 31 will be amended to provide that the application to be listed as a seller of poisons shall be made in duplicate and that one copy shall be endorsed by the local authority and returned to the applicant.

* * * * *

UNIVERSITY OF LONDON EXAMINATIONS IN VETERINARY SCIENCE

The Pass List for the University of London First Examination in Veterinary Science, for internal students, for December, 1948, intimates that the following were successful candidates: Tucker, Jean Winifred, and Wiltshire, Leslie Vasey, both of the Royal Veterinary College.

* * * * *

DISHORNING OF CATTLE

Amendment of the Animals (Anaesthetics) Act, 1919

For the information of readers whose attention it may have escaped, we report in this section the following notification recently made officially in our Notices column:—

The Minister of Agriculture and Fisheries and the Secretary of State for Scotland have made an Order (which came into force on January 1st, 1949) the effect of which is to permit cattle over one month old to be subjected to the operation of dishorning whilst under the influence either of a general anaesthetic or a local anaesthetic of sufficient power to prevent the animal from feeling pain.

* * * * *

AGRICULTURE (MISCELLANEOUS PROVISIONS) BILL

The Agriculture (Miscellaneous Provisions) Bill, the text of which was published on Thursday of last week, proposes the continuation of the livestock subsidy, and also provides other grants and loans to farmers.

As part of the Government policy for expanding livestock production a subsidy is now being paid for calf rearing and if—as it seems likely—this is continued for four years the cost will be approximately £30,000,000.

The calf-rearing subsidy is at the rate of £4 for a steer and £3 for a heifer, and is payable for calves born between August 21st, 1947, and October 1st, 1949, inclusive. The Bill also gives power to extend the payment of the subsidy, subject to the approval of Parliament, to September 30th, 1951. It is explained in the financial memorandum accompanying the Bill that the cost of £30,000,000 will be incurred if the subsidy is extended for a second two-year period and if the rates and conditions remain the same as in the 1947-49 period.

This Bill also authorises the provision or arrangement by the Government of facilities for training persons for employment in agriculture or horticulture. The annual expenditure on this is estimated at about £300,000 a year.

Another item of the Government's agricultural policy for which authority is provided in the Bill is the financial assistance being given by way of grant or loan—from August 21st, 1947, to March 31st, 1952—to schemes for the conservation of grass and forage crops. It is estimated that this assistance may amount to £800,000 in grants and to a similar amount in loans.

At an estimated cost of £250,000 authority is also given for a free (or reduced price) service from certain breeds of beef bulls at artificial insemination centres from October 1st, 1947, to March 31st, 1951.

ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

SHEEP SCAB:

Flints.—Ty Isa, Waen, St. Asaph (Dec. 31st).

* * * * *

JOINT N.V.M.A.-B.M.A. DIVISIONAL MEETING

Members of the Sussex and South Eastern Divisions were invited by the Hastings Division of the British Medical Association to attend a joint meeting held at the Queen's Hotel, Hastings, on Tuesday, January 4th, 1949, at 8.30 p.m.

Mr. S. L. Hignett, B.SC., M.R.C.V.S., gave an address on "Reproductive Disorders in Animals" which evoked a full and interesting discussion.

* * * * *

OLYMPIC GAMES AND INTERNATIONAL EQUESTRIAN FUND

In its January issue the *Horse and Pony News* records that at the meeting of the Council of the British Horse Society held on November 30th, 1948, Mr. E. Holland-Martin reported that the Trustees of the Fund, appointed by the British Horse Society and by the British Show Jumping Association, had held several meetings and were drafting a programme of activities for 1949, which would, they hoped, contribute towards the training of horses and riders for future Olympic Games. Jumping Teams were to be sent abroad to International Shows and it was hoped that it would be possible to assist with the expenses of the Three-Day Event and the Dressage Championship of the British Horse Society. An appeal for funds for this purpose had been circulated and it was hoped that members of the Council would bring the work of the Trustees of the Fund to the notice of those likely to give their financial support to its objects. A British Team had been sent to the recent Paris Show and had distinguished themselves with their successes in jumping competitions.

* * * * *

THE "HORSE AND HOUND" BALL will again be held at the Park Lane Hotel, London, on February 25th, 1949. Tickets, which cover light buffet refreshments, are £3 3s. double and £2 2s. single, and can be obtained from "Horse and Hound," The British Horse Society, and the B.S.J.A. This year the proceeds of this popular Ball are to be given to the Olympic Games and International Equestrian Fund.

* * * * *

At a recent meeting of the Council of the Royal Agricultural Society Sir William Dampier indicated that the Society's programme of research work was under review, but the Research Committee had had to realise that the time was ripe for new ground to be broken in view of the fact that their original work—making the bridge between research stations and farmers—had largely been taken over by the newly formed Agricultural Improvement Council, which had vastly greater resources than the Society, and could command expert services in every county. The significance of this was receiving the closest attention.

* * * * *

The term of office of a number of members of the University Grants Committee is about to expire and the committee has been reconstituted with the following membership: Sir Walter Moberly (chairman), Dr. A. E. Trueman (deputy chairman), Professor E. D. Adrian, Sir Charles Darwin, Miss D. Dymond, Mr. H. L. Elvin, Mr. E. James, Mr. H. S. Magnay, Professor W. R. Niblett, Professor P. S. Noble, Professor D. Hughes Parry, Professor G. W. Pickering, Professor E. K. Rideal, Professor H. G. Sanders, Professor J. C. Spence, Sir Alexander Murray Stephen, Mr. B. H. Sumner and Miss Barbara Wootton.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

Correspondents are requested to write as briefly as possible.

S. DUBLIN INFECTION IN ADULT CATTLE

Sir,—Through the excellent and comprehensive article on Salmonellosis by H. I. Field,[1] a disease entity has been brought to light which, though it has probably existed for many years in this country, has not until recently assumed serious proportions. Though much ground has been covered there are still some gaps in our knowledge of the subject which are baffling—the origin of the outbreaks, the essentially sporadic nature of each case and the absence of any direct association with disease in the human either before or after an outbreak in all but a few instances.

The symptoms and course of the disease are so well described by Field that only a brief reference is necessary here. There is very sudden and marked drop in milk yield, appetite is lost, the temperature quickly rises to something in the region of 106° F., within two days the animal commences to scour and fresh blood quickly appears and is often very marked by the fourth day. This blood is passed in large dark clots, which are very typical and have best been described by the owners of the animals as "like large pieces of rotting afterbirth." The course is very short, being frequently fatal in four to five days. We feel that it is desirable to draw further attention to this condition as its occurrence is becoming more common, especially in the west country. We have been prompted to write this letter by the discovery of a series of cases in a relatively small area in Dorset.

All the eleven outbreaks recorded in the table below occurred within a radius of three miles of a certain village and only adult stock over three years of age were affected. All cases occurred in August or September. There was no common water supply, no evidence of any human infection. No illness developed in hounds which were recipients of at least two carcases. In practically all our cases bulked milk from the whole herd, including that from the sick animal for the first two days, has been drunk raw by the farm personnel.

The theory that rats may be responsible for distributing the disease, for which Field has presented some positive evidence, may prove to be the correct one, or at least a part explanation, but we have not to date investigated this possibility.

The predisposing causes of the disease appear still to be obscure. We should have liked to discover some evidence of a nutritional deficiency in our patients which would have brought the situation into line with views expressed on nicotinic acid (P-P factor), by numerous workers quoted by Parry in his annotation on Nutrition and Resistance to Infection.[2] Therein it is suggested that "diarrhoeic signs attributed to nicotinic acid deficiency *per se* were in fact due to low grade intestinal Salmonellosis which the deficiency had permitted to develop." But in our cases victims were picked out at random from the milking herd without any previous warning of ill-health and without any signs of malnutrition as compared with their sisters.

One of us (Scott) has proved M. & B. 760 (sulpha-diazole) to be extremely valuable in the control of this disease. An initial dose of 75 grammes is administered as a drench in several pints of milk and water, and followed every twelve hours by 25 grammes similarly given until a total of 250 grammes has been received. So far no animal receiving this treatment has failed to recover, even when treatment has been introduced after the appearance of blood in the dung. Admittedly cases of spontaneous recovery do often occur, and have been observed by Field as well as ourselves, but in the outbreak recorded above symptoms were severe and the number of recoveries after treatment is, we feel, strong proof of the value of M. & B. 760 as a therapeutic agent.

Herd.		*Total No. of Cases.*	*Number Recovered.*	*Number Fatal.*
1.		1	0	1
2		1	1†	0
3.		2	1†	1
4.		1	0	1
5.	30*	2	1†	1
6		2	0	2
7.	36*	1	1	0
8.		1	0	1
9.	26*	3	2†	0
10.		1	0	1
11.		1	1	0
12		2	1†	1

* Number in milking herd.

† Treated with M. & B. 760.

Eleven cases received symptomatic treatment with astringents, but no M. & B. 760. Of these nine died and two recovered. All six cases which received M. & B. recovered quickly.

Yours faithfully,

N. S. BARRON, V.I.O., Reading.
D. C. SCOTT, M.R.C.V.S., Sherborne.

1 FIELD, H. I. (1948.) *Vet. J.* **104.** Nos. 8, 9, 10.
2 PARRY, H. *Vet. Rec.* **60.** 466.

DISPOSAL OF ANIMAL CARCASES

Sir,—May I endorse Mr. Kiddle's comments on the state of knackeries in this country.

Many veterinary practitioners must remember their first contacts with the revolting hovels which so often serve for the disposal of dead animals in the countryside; the familiar approach along a rutted road and the sudden vista of tin cans, rusty corrugated iron and in summer the disgusting stench of maggots and decaying meat. One's aesthetic senses are frequently shaken whilst learning the arts of veterinary surgery, but the first contact with a knacker's yard is probably one of the major shocks.

Anyone who has spent any time in Germany will realise that our methods of carcase disposal are both prehistoric and wasteful. Apart from being insanitary and an eyesore, knacker yards are responsible for a considerable waste of valuable raw material.

As the present state of affairs has existed for many years and there is little sign of active change in the industry, it does seem that it is up to the veterinary profession to accelerate improvement. This is a time of doubt about the success of nationalised industries, but there can be little hesitation in recommending some form of national policy for the disposal of dead carcases.

Whilst in Germany in the Control Commission, I was very impressed with the German system of carcase disposal as it seemed to conform with all requirements. It was usual to have a centralised factory which collected carcases daily from strategically placed collecting centres. At the factory the carcases were received on a large platform where they were then cut up and the various parts separated out. Bones went to be crushed and sterilised and the soft parts of the carcase were passed on to cutting and pulverising machinery and later sterilised. Most of the work was carried out mechanically and the final processed material appeared as a fine sterilised meal.

The notable features of this system were, that no part of the carcase was wasted, and also there was no danger of spread of disease as all the carcase material was sterilised by heat.

A very impressive feature, too, was that the carcases were carried to the factory in specially prepared lorry-borne containers. The containers resembled our milk float lorries or a large horizontal autoclave. As they were cylindrical and all metal, they could be readily disinfected. Extending from the top, at the end of the lorry, was a metal windlass which was used for pulling the carcases into the container, and, finally, the container was closed with a metal door which sealed the exit and prevented blood or infected material dropping on to the road. Such a type of vehicle affords a distinct contrast to the familiar knacker's lorry.

These factories and collecting centres were under veterinary control and were regularly inspected by the veterinary inspector of the Ministry. This system has many advantages as it cuts out waste, prevents the spread of disease and gives a useful guide as to the number of animals whose death is normally unrecorded. The centres, too, would provide very useful information on disease conditions.

It seems that this might be an opportune time to press for a definite improvement to be made in the present system of carcase disposal.

Yours faithfully,

The Cooper Technical Bureau, G. C. Brander.
Berkhamsted, Herts.
December 28th, 1948.

. . . . .

Sir,—I read Mr. Kiddle's letter on the above subject, in your issue of the 25th instant, with interest.

During the war I had occasion to work with a Netherlands veterinary surgeon and, early in our acquaintance, I asked him what struck him most about the difference between veterinary practice in this country and in his own. I was very surprised to find his answer, which came immediately, "Your, what do you call them, your knackers' yards. . . . Poof, how they smell!" This remark was vividly illustrated by a significant expression of nausea on his face. I understand that in the Netherlands the country is divided up into areas where Government controlled carcase disposal centres are situated, provided with motor transport for the collection of all animal carcases. These centres are equipped with electric skinning devices and proper facilities for *post-mortem* examinations, where they can be carried out under hygienic conditions and the by-products of the carcases efficiently salvaged. The latter remarks are, of course, hearsay, but these conditions certainly sound relatively ideal compared with those under which we have to carry out *post-mortem* examinations in this country.

I must confess—and I am sure I am not alone in this—that frequently, especially during the summer months, my stomach has rebelled at the thought of conducting a *post-mortem* examination where I have felt it should be done and consequently it has not been carried out. Nowadays, the animal carcase has much monetary value which could be available to provide these facilities and, if the centres were efficiently run, a higher price could be paid to the unfortunate owner, who loses a valuable beast.

Would it be possible for the N.V.M.A., together with the N.F.U., to instigate such centres?

It may appear paradoxical that the "doctor" should take an interest in the "undertaking business," but, from the practical point of view, it would have, I am sure, numerous advantages.

Yours faithfully,

The Olde Plough, Alex. A. Kidd.
Priest End, Thame, Oxon.
December 28th, 1948.

. . . . .

Sir,—*The Veterinary Record* of December 25th, 1948, published a timely letter by Mr. Kiddle on "Carcase Disposal Centres."

During the 1914-18 war when Germany was under the strain of a stiff blockade, the present idea for the disposal of condemned meat and carcases was adopted for the first time on a larger scale.

Since then, many improvements have been made and at present countries like Belgium or Holland have a country-wide utilisation scheme which can well serve as an example.

Wherever the idea of utilisation of animal carcases and condemned meat has taken root, the "knacker's yard," or, as Professor Burrow describes it (*J. roy. San. Inst.*, No. 2, March, 1947), the "black spot on the field of animal economy in this country," disappears. Its place is then taken by the disposal plant, a sanitary and industrial undertaking.

Britain's way of disposal of decaying, diseased and inedible carcases or parts of such falls short of present demands. Only a few districts can boast of properly organised removal of carcases and modern equipped utilisation plants. The demand for an all-out effort to check unnecessary waste and spread of disease is, therefore, indeed "worthy of comment and suggestions."

However, to develop this idea in the right direction, up-to-date legislation has to be passed, introducing a country-wide scheme. Supply districts have to be outlined and new disposal plants erected or existing ones modernised. The selected method of rendering should guarantee an efficient destruction of all disease-bearing organisms and give a favourable yield of the final products. The machinery should incur only moderate working expenses and demand little attention. Public ownership should be introduced, for the very same reason that the private abattoir had to give way to the public one. A full-time veterinary officer should be attached to each central plant, his duty being to examine carcases, report infectious diseases, supply valuable research material and supervise processing so that the final product, *i.e.*, the feeding-stuff, grease and fertiliser, is free from disease-carrying micro-organisms.

This would benefit national economy in two ways; first, by regaining valuable animal constituents, and secondly by protecting the health of the country's livestock by the destruction of dangerous infectious germs.

Yours faithfully,

A. Ginsberg.
Department of Veterinary Hygiene and Preventive Medicine,
Royal (Dick) Veterinary College, Edinburgh.
December 29th, 1948.

* * * * *

MEAT HYGIENE

Sir,—In the article on Meat Hygiene by Dr. Ginsberg and Professor Robertson, in the *Record* for January 8th, there appears a sentence which to my mind reads as though we were trying to put the clock back, as far as the handling of meat is concerned. After giving the "factory-type abattoir" a passing reference as being useful where meat is exported, the authors go on to suggest that the "German-type" abattoir would be appropriate for this country.

I would very respectfully beg to remind the two gentlemen concerned that animals have such things as hair, glands, offal, as well as two sides and a head. It is only in the factory-type abattoir that such material can be efficiently dealt with, and waste elimination carried out effectively.

Yours faithfully,

Brierley Hill, Staffs. D. J. Anthony.
January 8th, 1949.

Corrigendum.—"Is there a mare-gelding reaction?" (Questions and Answers, *Vet. Rec.* **61**, 14). In penultimate line of answer, *for* "umbilical" *read* "spermatic."